



UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
-----------------	-------------	----------------------	---------------------	------------------

09/724,319

11/27/2000

Dale B. Schenk

057437-391668

6653

826

7590

12/21/2010

ALSTON & BIRD LLP

BANK OF AMERICA PLAZA

101 SOUTH TRYON STREET, SUITE 4000

CHARLOTTE, NC 28280-4000

EXAMINER

BALLARD, KIMBERLY

ART UNIT

PAPER NUMBER

1649

MAIL DATE

DELIVERY MODE

12/21/2010

PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No. 09/724,319	Applicant(s) SCHENK, DALE B.	
	Examiner Kimberly Ballard	Art Unit 1649	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 08 March 2010.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 56-58,61,63-66,71-79,81,85,86,92-94,97,99,164-191 and 194-217 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 56-58,61,63-66,71-79,81,85,86,92-94,97,99,164-191 and 194-217 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
 Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
 Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. _____.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--|---|
| 1) <input type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413) |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | Paper No(s)/Mail Date. _____ |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| Paper No(s)/Mail Date <u>03/08/2010 (6)</u> | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

Status of Application, Amendments, and/or Claims

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on March 8, 2010 has been entered.

2. Claims **56-58, 61, 63-66, 71-79, 81, 85, 86, 92-94, 97, 99, 164-191** and **194-217** are pending and under consideration in the current office action.

Information Disclosure Statement

3. The six information disclosure statements (IDSs) filed on March 8, 2010 have been considered and the references therein are of record.

Withdrawn Claim Rejections

4. The rejection of claims 56-58, 61, 63-66, 71-79, 81, 86, 92-94, 97, 99, 164-191, 194-203, 205, and 207-209 under 35 U.S.C. 103(a) as being unpatentable over U.S. Patent 5,589,154 to Anderson in view of EP 613007 to Becker and U.S. Patent 5,593,846 to Schenk, is withdrawn in view of Applicant's arguments at pp. 13-14 of the response and the declaration under 37 CFR 1.132 by Dr. John Anderson, from co-

Art Unit: 1649

pending Application No. 10/923,471. In particular, the declaration discusses the difficulties that would have been encountered at the time of filing of the instant invention (the late 1990s) with respect to the technique for amino acid sequencing of an antibody, and how the conventional means of making a humanized antibody could not have been performed without access to the hybridoma.

5. The Declaration under 37 CFR 1.132 filed October 8, 2009 (originally filed in co-pending Application No. 10/923,471; the Declaration by Dr. Seubert) is sufficient to overcome the rejection of claims 57, 99 and 184 based upon the US Patent to Schenk (US 5,593,846) applied under 35 U.S.C. 103(a). In particular, the declaration indicates that the research community's access to the hybridoma that produces the 266 antibody and use of the antibody itself would have been restricted to research purposes only. Exemplary material transfer agreements to this effect have also been provided as in the response filed October 8, 2009.

6. The rejection of claims 85 and 204 under 35 U.S.C. 103(a) as being unpatentable over US Patent 6,303,567 to Findeis et al. in view of Solomon et al. (*Proc Natl Acad Sci, USA*, Jan 1996; 93:452-455) and EP 613007 by Becker, and further in view of EP 0620 276 A1 by Adair et al., set forth at paragraph 15 of the previous office action mailed 04/08/2009, is withdrawn in view of Applicant's arguments at pp. 19-20 of the October 8, 2009 response, in view of Hogarth reference (*Curr Opin Immunol.* 2002; 14:798-802), and in view of the declaration of Shyra Gardai (originally filed April 3, 2009

Art Unit: 1649

in co-pending Application No. 09/322,289). Based upon the references of record, in this instance it would not have been obvious to select a human, chimeric or humanized antibody of the IgG1 isotype.

Maintained Claim Rejections

Double Patenting

7. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the “right to exclude” granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

8. The nonstatutory provisional obviousness-type double patenting rejection of claims 56-58, 61, 63-66, 71-79, 85-86 and 92-94 over claims 164-184, 187-207, 210-217, 220-221, and 223-226 of copending Application No. 10/923,469 is maintained for reasons of record and held in abeyance until all other rejections are resolved.

Art Unit: 1649

9. The nonstatutory provisional obviousness-type double patenting rejection of claims 97, 99, and 164-182 over claims 164-166, 168-179, 185, and 187-193 of copending Application No. 10/923,471 is maintained for reasons of record and held in abeyance until all other rejections are resolved.

Claim Rejections - 35 USC § 103

10. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

The factual inquiries set forth in *Graham v. John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

11. Claims 56, 58, 61, 63-66, 71-76, 81, 86, 92-94, 97, 164-183, 185-191, 194-199, 203, 205, 207-209 and 212-215 are rejected under 35 U.S.C. 103(a) as being unpatentable over US Patent 6,303,567 B1 to Findeis et al. (issued October 16, 2001, filed August 27, 1996; listed on IDS filed 04/27/2004) in view of Solomon et al. (*Proc Natl Acad Sci, USA*, Jan 1996; 93:452-455; listed on IDS filed 08/20/2001) and EP

613007 by Becker (published August 31, 1994; of record). The rejection is maintained for reasons of record and as set forth below.

Response to Arguments

12. In the response filed October 8, 2009, Applicant argues that impermissible hindsight was used in this rejection. In particular, Applicant asserts that the art as a whole at the relevant time did not point to the 17-20 or 21 region of A β as being the key region to focus therapeutic treatments. Applicant points to WO 95/08999 (cited as reference 531 on IDS filed 04/29/2005), which Applicant alleges teaches away from the notion that residues 16-20 are an effective anti-aggregating epitope for targeting therapeutics. In this publication, administration of the peptides A β 16-20 and A β 16-22 had no effect in a mouse model of Alzheimer's disease, and therefore Applicant argues would have suggested that these peptides were ineffective at inhibiting aggregation of A β *in vivo*, thus teaching away from attempts to develop compounds binding to such epitopes to inhibit aggregation.

Applicant also asserts that Becker implicitly teaches away from using an antibody directed to a 17-20 or 21 epitope, because Becker proposes A β toxicity results from the beta-pleated conformation of β -amyloid, and Barrow (*J Mol Biol.* 1992; 225(4):1075-1093) reports that residues 29-42 of A β are most associated with the β -sheet conformation. Based upon Barrow, therefore, Applicant argues that one of skill in the art following Becker's proposal would have selected an antibody directed to the 29-42 region. In addition, Applicant alleges that Becker does not unambiguously disclose an intent to administer antibodies that bind to the random coil form of A β (as opposed to

the beta-pleated sheet form) therapeutically or provide any reason to think they would be effective therapeutically.

At pp. 16-17 of the response, Applicant cites several prior art references that teach various portions of the A β peptide as being involved in A β -mediated neurotoxicity and/or as potential targets for therapeutic applications. Applicant notes that “although not all of these references negate A β 17-20 or 17-21 as having a possible role in aggregation, they do teach away from it being the only such epitope or critical epitope for therapeutic efficacy and set the skilled artisan on a divergent path from the claimed methods.” When view in the aggregate, Applicant argues, a skilled person could not have confidently identified which region of A β was critical for mediating aggregation or toxicity.

Applicant further asserts that small molecules and antibodies are not equivalents. Given the difficulty in treating Alzheimer's disease at the time of filing and the surprise of experts in the field when methods consistent with the presently claimed method elicited beneficial effects in transgenic AD mice, Applicant argues that it would not have been predictable to substitute antibodies for small molecules to obtain successful treatment of AD. Applicant asserts that there were not a finite number of identified predictable solutions for treating AD, and that there was unpredictability with respect to the development of effective therapeutics. Again Applicant argues that impermissible hindsight rather than the common sense approach was used, and the references do not provide a reasonable expectation of success.

Art Unit: 1649

13. Applicant's arguments have been fully considered but they are not persuasive. In response to applicant's argument that the examiner's conclusion of obviousness is based upon improper hindsight reasoning, it must be recognized that any judgment on obviousness is in a sense necessarily a reconstruction based upon hindsight reasoning. But so long as it takes into account only knowledge which was within the level of ordinary skill at the time the claimed invention was made, and does not include knowledge gleaned only from the applicant's disclosure, such a reconstruction is proper. See *In re McLaughlin*, 443 F.2d 1392, 170 USPQ 209 (CCPA 1971).

With respect to WO 95/08999, the examiner has closely examined the sections of the prior art reference that Applicant states are indicative of the non-obvious nature of the specific epitope consisting of residues 16-20 of A β . Applicant argues that WO 95/08999 teaches away from the claimed invention. This reference does not teach away from the claimed invention. The reference discloses that a short peptide, consisting of residues 16-20 of A β , is not therapeutic when administered to animals with experimentally induced Alzheimer-like pathology. This does not show that *antibodies* against residues 16-20 would be ineffective, which is what is claimed, it merely shows that a peptide consisting of residues 16-20 was itself not therapeutic in the regime administered. The animals were tested one week after administration of the peptides, which is likely not sufficiently long for a therapeutically effective antibody response to develop. For a therapeutically effective immunization protocol, the present specification discloses that relatively large doses (up to 500 μ g per human patient, sometimes as high as 1-2 mg) of antigen are to be used, and a booster injection is typically re-

Art Unit: 1649

administered about 6 weeks later (see pp. 27-28). In the WO document, only a single dose was administered intracerebroventricularly (i.c.v.) one week prior to testing; not only is this too short a period of time to have had antibodies develop, it would not be expected that an antibody response would be elicited when an agent is administered directly into the brain. Thus the finding that residues 16-20 or 16-22 of A β are not anti-amnesia drugs does not speak to the non-obvious nature of the presently claimed invention, which is a method of administering antibodies. Because WO 95/08999 only reports the results of an experiment when this peptide was administered i.c.v. for a short period of time, it is improper to conclude that antibodies directed against the same peptide would be ineffective for treating Alzheimer's disease.

In fact, contrary to Applicant's assertions, WO 95/08999 is on point to selection of an antibody that recognizes an epitope within residues 13-28 of A β as claimed because the WO document teaches that amyloid-like fibrils arise readily *in vitro* under physiological conditions even for the smaller A β peptides of A β 1-28, A β 12-18 and A β 18-28, wherein extensive stacks of β -pleated sheets are formed from the latter peptide (i.e., A β 18-28) (see p. 3 of document). The WO document used the A β 12-28 peptide to induce amnesic effects in test mice, which they noted is a peptide that is as potently amnesic as A β and which shows amyloid-like aggregation similar to A β . These teachings would indicate that the researchers construed this A β 12-28 fragment as representative of a core molecule responsible for amyloid pathology, and that inhibition of this peptide's negative effects would be therapeutically beneficial.

Applicant also argues that Becker implicitly teaches away from using an antibody against these specific residues. According to Applicant, Barrow et al. teach that a different region of the A β peptide, namely residues 29-42, are most associated with β -sheet conformation. Applicant argues that this would lead one of ordinary skill in the art to pursue that region, not residues 17-20 or 17-21, as a possible therapeutic. The examiner disagrees with Applicant's characterization that this constitutes even an implicit teaching away from the claimed invention. Nothing in either Barrow or Becker suggests that residues 29-42 of A β are the only region that should be targeted, and that no other region is a possible candidate for therapy. While Barrow may indicate that residues 29-42 are the first to aggregate, this does not suggest that this is the only region that is important to fibril formation. Quite the contrary, as noted above, WO 95/08999 indicates that residues 12-28 of A β are involved in amyloid aggregation and pathology, and Findeis et al. guides one of ordinary skill in the art directly to the core sequence of residues 17-20 or 17-21 of A β as a fibril-forming region. That multiple possible fibril-forming epitopes were known in the art does not speak to the non-obviousness of residues 17-20 or 21 in particular. See MPEP § 2145(X)(D)(1).

Additionally, Applicant asserts that the thrust of Becker's disclosure is directed to the correlation between β -sheeted structure and toxicity of A β , such that Applicant alleges the skilled artisan would not assume that the other conformationally-specific antibodies disclosed by Becker, those which bind to random coil A β , are intended to be used therapeutically. Applicant's arguments, however, are not persuasive. Becker does indeed teach antibodies that bind predominantly to either the β -sheet conformation or

Art Unit: 1649

the α -helical (random coil) conformation of β -amyloid, but indicates that both types of antibodies are appropriate for therapeutic or diagnostic use. At column 7 lines 26-38, Becker teaches both forms of the antibodies. The next paragraph describes the uses of the antibodies, and indicates that the uses include "... in vivo administration to mammals, preferably humans" (column 7, lines 47-52, emphasis in original). At column 8, lines 16-18, Becker also teaches that "[t]he antibodies of the present invention are useful in the diagnosis and treatment of mammals suffering from Alzheimer's disease." Note that the last two statements quoted above, each of which discusses administration of antibodies for treatment of Alzheimer's disease, are *generic* with respect to the antibody to be administered. Becker refers to "[t]he antibodies of the present invention" (column 7, lines 49-50 and column 8, line 16), meaning all the antibodies disclosed therein, and states that they are effective for both diagnostic and therapeutic uses. Much as Becker defined the term "antibody" to include humanized antibodies and antigen-binding fragments, he also defined the term to include both antibodies that bind predominantly to α -helical forms of $A\beta$ and those that bind predominantly to β -sheet forms of $A\beta$. One of skill in the art would clearly understand that Becker considered both types of antibodies appropriate for therapeutic applications.

Applicant also discussed the references by Frenkel, Yankner et al., Velazquez et al., and Giulian et al. as evidence that other regions of $A\beta$ may play important roles in the disease process, possibly guiding one of ordinary skill in the art to other regions of the peptide as epitopes to which antibodies should be raised. Again, although other regions of $A\beta$ were recognized as possibly being involved in the generation of

Art Unit: 1649

aggregates or fibrils, nothing in the references cited teaches away from residues 17-20 or 17-21 as being an important region for this toxic phenomenon. Since Findeis explicitly teaches that this region should be inhibited, one of ordinary skill in the art would have found it obvious to apply the methods of Becker using this specific knowledge, thereby arriving at the claimed invention. And while it is true that small molecules and antibodies do differ with respect to size and bioactivity, the general suggestion that the ordinary skilled artisan would be left regarding the Findeis reference is that agents which *bind to* the core fibrillogenic sequence of A β , which comprises residues 17-21, would be therapeutically beneficial for inhibiting A β aggregation and A β neurotoxicity. Even apart from the teachings of Becker and Solomon, one of skill in the art would have recognized antibodies as agents that are specialized for binding to particular antigenic epitopes

With respect to the argument that there were not a finite number of identified predictable solutions for the treatment of Alzheimer's disease, this alone does not mean that the presently-claimed invention would have been non-obvious. Certainly, many fields are characterized by a plethora of different approaches for solving a specific problem, nonetheless the presence of such a diversity of possible solutions does not indicate that a particular one is non-obvious. In the present case, the references of record directly guide one of ordinary skill in the art to perform the method now claimed. Findeis teaches modulator compounds that bind to A β and inhibit the neurotoxicity of A β fibrils, particularly those compounds which bind A β 17-20 or A β 17-21. Becker teaches administration of antibodies that bind to certain forms of A β so as to inhibit A β

Art Unit: 1649

neurotoxicity, including humanized antibodies. Solomon teaches that inhibition of A β aggregation is useful therapeutically, and suggests the use of monoclonal antibodies and single chain antibodies to inhibit A β aggregation so as to reduce neurotoxicity for the treatment of Alzheimer's disease. Given these explicit teachings, one of ordinary skill in the art would have found the selection of antibodies that bind to the specific epitope that consists of residues 17-20 or 17-21 of A β obvious. The prior art thus provides a reasonable expectation of success that administration of a compound, such as an antibody, that binds to the region comprising this fibrillogenic epitope will be therapeutically beneficial because it will inhibit A β aggregation and/or A β -mediated neurotoxicity.

14. Claims 77-79 and 200-202 are rejected under 35 U.S.C. 103(a) as being unpatentable over US Patent 6,303,567 B1 to Findeis et al. in view of Solomon et al. (*Proc Natl Acad Sci, USA*, Jan 1996; 93:452-455) and EP 613007 by Becker as applied to claims 56, 58, 61, 63-66, 71-76, 81, 86, 92-94, 97, 164-183, 185-191, 194-199, 203, 205, 207-209 and 212-215 above, and further in view of Plückthun (*Immunol Rev.* 1992; 130:151-188). The rejection is maintained for reasons of record.

Response to Arguments

15. In the response filed October 8, 2009, Applicant traverses the rejection at least for the reasons already given in connection with Findeis, Solomon and Becker above.

16. Applicant's arguments have been fully considered but they are not persuasive. Applicant's arguments with respect to the Findeis, Solomon and Becker references

Art Unit: 1649

have been discussed above, and therefore the rejection of claims 77-79 and 200-202 is maintained.

17. Claims 210, 211, 216 and 217 are rejected under 35 U.S.C. 103(a) as being unpatentable over US Patent 6,303,567 B1 to Findeis et al. in view of Solomon et al. (*Proc Natl Acad Sci, USA*, Jan 1996; 93:452-455) and EP 613007 by Becker as applied to claims 56, 58, 61, 63-66, 71-76, 81, 86, 92-94, 97, 164-183, 185-191, 194-199, 203, 205, 207-209 and 212-215 above, and further in view of Trang et al. (*Pharm Res.* 1990; 7(6):587-592). The rejection is maintained for reasons of record.

Response to Arguments

18. In the response filed October 8, 2009, Applicant traverses the rejection at least for the reasons already given in connection with Findeis, Solomon and Becker above.

19. Applicant's arguments have been fully considered but they are not persuasive. Applicant's arguments with respect to the Findeis, Solomon and Becker references have been discussed above, and therefore the rejection of claims 210, 211, 216 and 217 is maintained.

Conclusion

20. No claims are allowed.

21. All claims are drawn to the same invention claimed in the application prior to the entry of the submission under 37 CFR 1.114 and could have been finally rejected on the

Art Unit: 1649

grounds and art of record in the next Office action if they had been entered in the application prior to entry under 37 CFR 1.114. Accordingly, **THIS ACTION IS MADE FINAL** even though it is a first action after the filing of a request for continued examination and the submission under 37 CFR 1.114. See MPEP § 706.07(b). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Advisory Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Kimberly Ballard whose telephone number is 571-272-2150. The examiner can normally be reached on Monday-Friday 8:30 AM - 5 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Stucker can be reached on 571-272-0911. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Kimberly Ballard
Art Unit 1649

/Elizabeth C. Kemmerer/
Elizabeth C. Kemmerer, Ph.D.
Primary Examiner, Art Unit 1646